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DISEASE OF THE BRAIN

IN ITS RELATION TO

INFLAMMATIONS OF THE EAR.

BY

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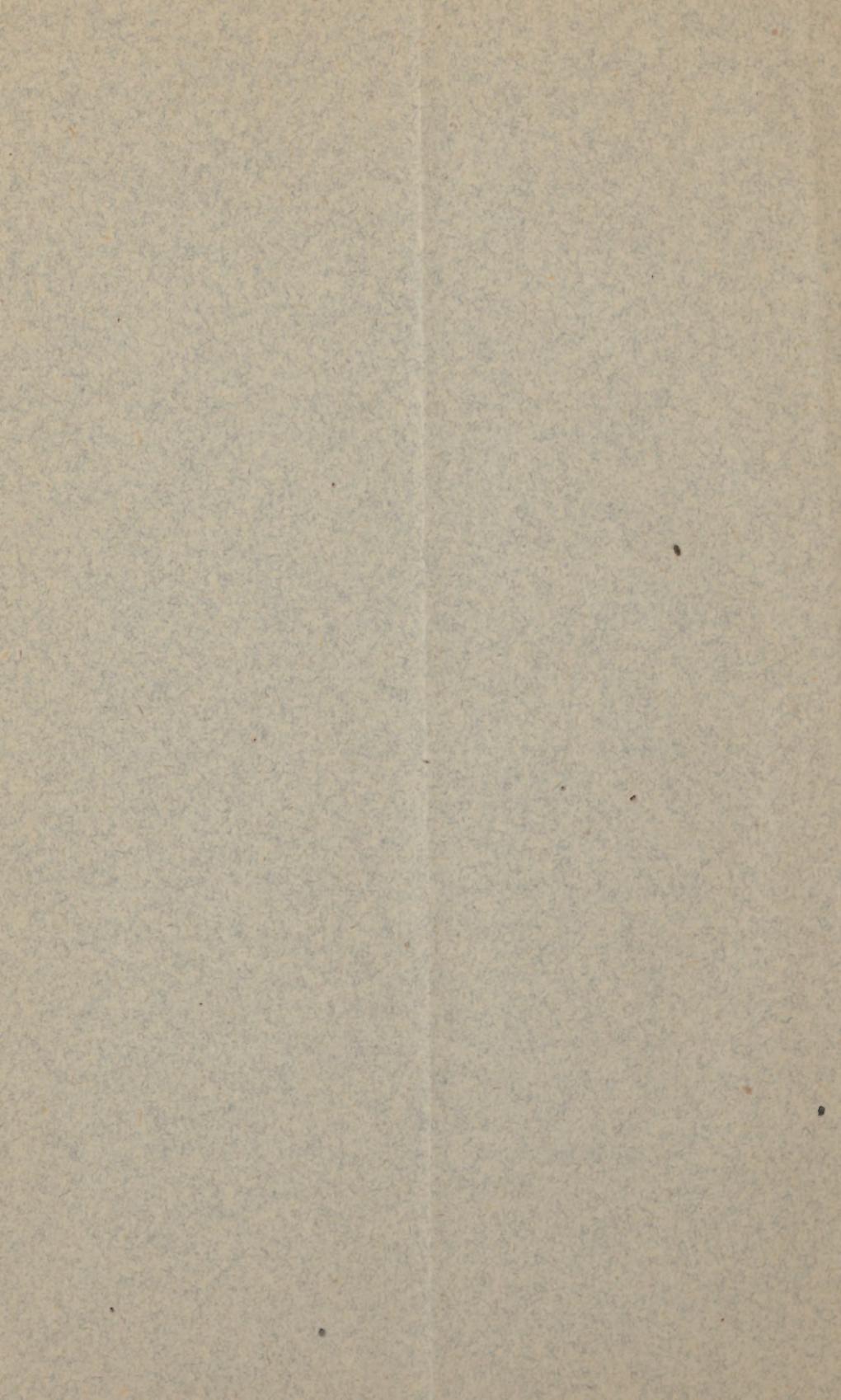
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That a purulent discharge from the ear might occasionally cause necrosis of the bone in which that organ is imbedded, and that this necrosis might cause fatal disease of the brain has been recognized for a very long time by pathologists. That such a purulent otitis might become the direct cause of death without the bone becoming diseased has been known only of late years. The dissection of pathological specimens has shown, however, that necrosis is not the only thing to be feared in these cases, but that, even if the petrous bone is unaffected, the disease of the ear may be the direct cause of death.

The anatomical relations of the ear, as shown by the most recent investigations, prove that the ear is in more intimate connection with the brain and other important parts than had been supposed. By means of the microscope and fine injections it has been shown that the numerous small foramina with which the temporal bone is perforated furnish passages through which an inflammation may extend to other parts.

Troeltsch says, "Not only the true diplöe, but the bone of the os temporis in general, is in direct connection, by means of its blood-vessels, with the dura mater on the one hand and with the soft parts of the ear on the other. The temporal bone in general receives its blood-vessels from within and from without, and also sends them in both directions, not only to the dura mater, but also to the membranes lining the outer and middle ear. Diseases of the latter produce abnormal conditions in the bone and its vessels, which, either through the contents, or along the tissue of the

walls of the blood-vessels, pass on to the dura mater, and there cause secondary pathological processes. These announce themselves in the one case by purulent inflammations of the brain membranes, or of the walls of the sinuses; in another, by clot formation and closure of the calibre of the vessels, or by the entrance of putrid matter into the circulation. That all of these processes, developing themselves within or on the vessels, can be produced by the purulent inflammation of the soft parts of the ear without the existence of a 'caries of the petrous bone,' cannot often enough be impressed upon the practitioner, since many are inclined to fear only a caries of the petrous bone, not, however, a simple otorrhœa or purulent inflammation of the soft parts of the ear."

The whole upper and inner surfaces of the petrous bone lie in direct contact with the meninges of the brain, being covered with the dura mater which here serves as a periosteum and nourishes the bone. Part of the upper surface forms the roof of the tympanum, is variable in thickness, but frequently so thin as to be nearly transparent, and occasionally is partially wanting from congenital defect.

The lateral sinus, a fold of the dura mater, and like it serving as a periosteum, is only separated from the mastoid cells by a thin lamella of bone, through which often many minute foramina pass directly into the cells which constitute part of the tympanic cavity. The posterior temporal vein of the diplœ of the skull enters the lateral sinus. In addition to this the mastoid foramen serves for the passage of a vein from the scalp into the lateral sinus, so that through these two veins we have the venous circulation of the diplœ and of the scalp in direct connection with that of the brain. The facial nerve in its Fallopian canal passes directly through the tympanum, is never separated from that cavity by more than a very delicate, bony plate, and frequently from a deficiency in this plate, probably due to an arrest of ossification, lies in direct contact with the mucous membrane lining the tympanum.

The floor of the tympanum is formed by the jugular fossa, in which lies the internal jugular vein; in this floor is a foramen through which a branch of the vagus passes into

the tympanum. Occasionally at this spot also, from an arrest of ossification, the coverings of the jugular vein lie in direct contact with the mucous membrane of the tympanum.

The anterior wall of the tympanum is formed by the carotid canal, is so thin that light passes through it readily, and is, moreover, perforated by foramina, through which the tympanic branches of the sympathetic pass from the carotid plexus to the tympanum.

The meatus internus gives a large canal from the cavity of the skull to the labyrinth of the ear, and this latter is separated from the tympanum only by the thin membranes covering the fenestræ, ovalis and rotunda, through which the blood-vessels of the tympanum and labyrinth communicate by anastomoses. This large passage is lined by a prolongation of the dura mater, which serves as its periosteum. The aquæductus vestibuli also connects the interior of the skull with the cavity of the labyrinth, and serves for the passage of a small vein.

The petrosal-mastoid canal leads from the mastoid cells to the interior of the skull, thus furnishing still another communication from a different part of the tympanum to the brain. It serves for the passage of a vein which has been followed into the superior petrosal sinus, so that we have the circulation of the tympanum here in direct communication with that of the meninges of the brain.

When, in addition to these anatomical relations and connections, it is remembered that the mucous membrane which lines the whole tympanum is so intimately connected with the periosteum that the two cannot be distinguished, or, in other words, that the mucous membrane of the tympanum constitutes, in fact, the periosteum on the interior of the petrous bone, the only wonder is that disease of the brain is not a more frequent sequel of inflammation of the ear than it is.

The inflammations of the ear from which, as experience teaches, disease of the brain arises, consist essentially of a suppurative inflammatory process of the mucous membrane which lines the whole tympanic cavity;—in fact, a periostitis of the interior of the petrous bone.

It is unnecessary here to speak of the symptoms and appearances of a purulent inflammation of the tympanum; we are concerned only with the fact that, as the result of this inflammation we have the tympanic mucous membrane intensely inflamed, secreting pus, often in large quantities, and with a very great tendency to ulceration. The purulent inflammation of the tympanum generally involves the mucous membrane of the whole cavity; that is, both the tympanum proper and the mastoid cells. But this is not necessarily the case; sometimes the inflammation is confined to the tympanum proper, and a few rare cases have been reported in which the mastoid cells alone were involved.

The usual course of such an inflammation is as follows: the pus secreted in the tympanum gradually fills that cavity, ruptures through the drum-membrane and discharges itself into the external meatus. This evacuation of the pus is followed, in favorable cases, by a subsidence of the inflammation of the mucous membrane, a gradual cessation of the secretion, healing of the perforation and a restoration to the normal condition of all parts of the tympanum. If the mastoid cells are involved they also become filled with pus; but, as the inflammation of the tympanum proper subsides after rupture of the drum-membrane, the inflammation of the cells also subsides and this pus is absorbed. The deviations from this usual course of the disease are: retention of the pus in the tympanum proper, owing to abnormal thickening of the drum-membrane, which resists rupture, or owing to the rupture being insufficient to thoroughly evacuate the cavity; affection of the bone in any part from the inflammation of the mucous membrane which serves as periosteum; extension of the inflammation along some of the channels of communication already mentioned.

While the usual course of such an inflammation, even when complicated as just described, is favorable, as far as the brain is concerned, there are certain risks to which that organ is exposed; namely, (1) the danger of an extension of the inflammation *per continuitatem* along some of the avenues of communication already described, this extension of the inflammation being aided by the pressure of the pus confined

in the bony cavity ; (2) the danger of invasion of the neighboring tissues from extension of the inflammation *per contiguitatem* ; (3) the danger of a decomposition of the confined pus, causing phlebitis in any of the veins communicating with the brain. As the result of the two former dangers we may have an ostitis of the bone, caries or necrosis, either very circumscribed or extensive, or a circumscribed inflammatory softening and absorption of the bone.

The diseases of the brain which follow inflammation of the ear are either meningitis, encephalitis, phlebitis or thrombosis of the sinuses or jugular vein.

Acute meningitis may result from an extension of the inflammation of the tympanum *per continuitatem* along the minute blood-vessels which connect the circulation of the ear with that of the brain ; or it may result from the direct irritation of a caries either of the roof of the tympanum or of the inner wall of the mastoid, or from the rarer cases of caries of the whole petrous bone. The penetration of the purulent matter into the labyrinth by ulceration or rupture of the small fenestrae, and thence along the meatus internus or aquæductus vestibuli, is also a not infrequent cause of meningitis.

The meningitis caused by an inflammation of the ear does not differ essentially in its anatomical features from that arising from any other cause : it may extend over the whole surface of the cerebrum, or be limited to the surface in the neighborhood of the diseased ear, or, more commonly still, it is confined to the base of the brain. Its connection with the ear, although not always conspicuous, can generally be traced by examining carefully the points at which the ear communicates with the brain. It will often be found that in the neighborhood of the petrous bone the congestion is more marked than in other parts of the meninges ; often that, in this neighborhood, the inflammation is in a more advanced stage than over the rest of the meninges ; that while over the general surface of the base of the brain or cerebrum the inflammation is in the early stage of congestion, near the petrous bone it has already gone on to the deposit of lymph, or has become purulent. After the removal of the brain, an

examination of the petrous bone will often establish most satisfactorily the source of the inflammation; for the dura mater over the bone will be found intensely inflamed at the spot of communication with the ear, and not infrequently an incision will develop a minute quantity of pus lying between the dura mater and the bone. The points at which this is most likely to be the case are either the upper surface of the petrous bone, over the roof of the tympanum, or the orifice of the aquæductus vestibuli on the posterior aspect of the bone between the lateral sinus and the meatus internus, or the meatus internus. In the cases where the ear has become more seriously diseased, the connection of the meningitis with that organ is generally so marked that it can scarcely escape even a superficial examination. If the petrous bone has become carious, or been perforated, there is generally a well-marked deposit of lymph or pus around the diseased bone, and often the cerebral surface of the dura mater is so firmly attached to the bone that it can only be separated with difficulty. Sometimes the dura mater over the diseased bone is found perforated from ulceration. By stripping the dura mater from the bone, any inflammation, perforation, or caries of the bone is to be seen distinctly. The congenital defect in the roof of the tympanum which is occasionally found would, of course, favor the extension of an inflammation from the ear to the meninges, as the dura mater would then lie in direct apposition with the mucous membrane of the tympanum.

In some cases, in which death has been caused either by some other disease of the brain than meningitis, or by some general disease, it is interesting to see how the brain has been protected from injury even where the petrous bone has become carious to a great extent. As the result of a slow, long-continued inflammation of the ear and petrous bone, the dura mater over the bone becomes greatly thickened and affords a firm barrier to the extension of the inflammation. On this account a chronic inflammation of the ear, extending over years without acute exacerbations, is less to be feared than one which, however long continued, takes on suddenly an acute character from exposure or some other cause, and

subjects the patient to the risks of an increased pressure of pus and renewed ulceration. In these cases where the meninges seem to be protected by the thickening of the dura mater, and yet a fatal meningitis has been set up by the ear, it will not infrequently be found that the acute exacerbation has caused a purulent inflammation of the labyrinth, and this has extended to the meninges through the meatus internus along the auditory nerve.

Whether disease of the ear is ever the cause of a chronic meningitis is uncertain. I have never seen it spoken of as such, and am not aware of any observations which point toward such a connection between an inflamed ear and the opaque, thickened arachnoid found in chronic meningitis.

Phlebitis and thrombosis as the result of inflammation of the ear may occur primarily either in the sinus lateralis, the sinus petrosus superior, the sinus petrosus inferior, or in the internal jugular vein. The close relation and connection of these veins with the tympanum have been already mentioned.

The lateral sinus is liable to inflammation from an extension of the mastoid inflammation along the minute blood-vessels which supply the bone between the cells and the sinus, and which fill the minute foramina observed on a macerated bone; or it may become inflamed from a caries of the bone on the inner wall of the mastoid cells; or from an inflammation of the bone of the mastoid (ostitis), which, by causing a phlebitis of the veins of the diploë, would by extension affect the lateral sinus through the chief vein of the diploë, already mentioned.

The superior petrosal sinus may become inflamed from an extensive caries of the petrous bone, or from a circumscribed inflammation or caries of the tympanic roof, or finally from an extension of the inflammation of the mastoid cells along the petrosal-mastoid canal, which conveys a vein from the mastoid cells directly into the petrosal sinus. It may be affected secondarily from an extension of a thrombus or phlebitis of the lateral sinus.

The inferior petrosal sinus, although not in direct connection or relation with the ear, may become inflamed from an extensive caries of the petrous bone, or from an inflammation

or caries around the orifices of the aqueductus vestibuli or meatus internus, or secondarily by extension of the disease from the other sinuses.

The jugular vein is liable to inflammation from the same causes as the lateral sinus, although not so frequently affected as the sinus. The tympanic inflammation may extend along the minute foramina in the bone, which gives passage to minute nerves and blood-vessels, and thus cause a phlebitis of the jugular; or the bone between the tympanum and the jugular fossa may become carious; or finally there may be a congenital defect in the bone at this point which places the inflamed mucous membrane of the tympanum in direct apposition with the coverings of the jugular vein. Finally, like the sinuses, the jugular may be affected secondarily from an extension downwards of an inflammation in the lateral sinus.

Encephalitis from disease of the ear is always found, in its most advanced stage, as abscess of the substance of the brain. The position of the abscess varies very much, and in some cases its connection with the ear-disease is by no means easily made out. The most frequent position of the abscess is in the cerebral hemisphere next the diseased ear; it may be on the lower surface of the hemisphere directly over and communicating with a caries of the tympanic roof; or it may be in the substance of the hemisphere, with a portion of healthy brain-tissue between it and the diseased bone; or, in very rare cases, it may be in the substance of the cerebral hemisphere on the opposite side from the ear-disease. The next most frequent position of the abscess, after the cerebrum on the side of the ear-disease, is the cerebellum on the side of the ear-disease; and, as in the cerebrum, the abscess may be here on the lower surface, or in the substance of that organ. These abscesses may be either single or multiple, and in the latter case they may all be in one hemisphere, or they may be in different parts of the brain.

Meyer,* in his exhaustive monograph on the pathology of abscess of the brain, gives an excellent description of encephalitis from otorrhœa. He reports that of 89 cases of suppuration of the brain recorded by Lebert, 20 were caused by

* Zur Pathologie des Hirn-Abscesses von Dr. Rudolph Meyer. Zurich, 1867.

otorrhœa; of 40 cases of Schott's, 13 were due to otorrhœa, while of his own, 86 cases, 20 were caused by otorrhœa. This gives of 206 cases of abscess, 53 from disease of the ear, which is nearly the proportion caused by injuries, as an analysis of the same statistics shows that in one-fourth of the cases the abscess was the result of injury of the head. Suppuration of the cerebrum occurs very frequently (10 times in 19 cases), and the right hemisphere is its most frequent seat (9 times in the 10 cases), as would be expected from the fact that the right petrous bone was found more frequently diseased than the left one (14 cases, right : 4 cases, left). Whether otorrhœa is more frequent on the right side than the left is unfortunately not known; but it should be stated that several observers have already called attention to the above fact. In only two cases did multiple abscesses follow caries of the right petrous bone. In the first of these cases one abscess was in the cerebrum and the other in the cerebellum; in the second case there were three abscesses in the cerebellum. Usually, it is only a chronic otorrhœa which leads to suppuration of the brain; for of nineteen cases there was only one which arose from an acute otitis. Not infrequently a chronic catarrh of the mucous membrane of the external meatus or of the tympanum causes a polypoid hypertrophy of the mucosa on the one surface, and a chronic inflammation of the neighboring bone on the other surface. The caries of the petrous bone, thus produced, causes an inflammation and close adhesion of the dura mater, and from here the inflammation glides into the deeper parenchyma of the brain, sometimes in such a way that the dura mater forms the anterior covering of the abscess, sometimes so that the abscess remains concealed deep in the parenchyma. Frequently, fistulae exist between the abscess in the brain and the suppurating bone; but this is not always the case: it is by no means necessary that the dura mater and the bone should be in any way defective. Gull calls attention to the fact that perfectly healthy brain-tissue may lie between the abscess and the diseased bone, and that then we must suppose that the abscess of the brain occurs in the way that it does, not infrequently, from inflammation of still more

distant parts. This origin of the abscess by metastasis from an otorrhœa is by no means impossible, yet it is certainly exceptional; at least, in Meyer's cases there are no examples of the abscess being on the opposite side from the ear disease.

In a case of caries of the petrous bone it is not always possible to distinguish between a thrombus of the lateral sinus and an abscess of the brain; the two diseases combined are found in one of Meyer's cases, where the bone on the posterior surface of the petrous bone was diseased and had given rise to polypoid excrescences on the anterior wall of the sinus. The seat of the abscess in the brain depends in a great measure on the position of the affection of the bone, and quite regular laws can be established, as Toynbee first showed. According to him the retention of the otorrhœal products is the chief cause of the extension of the inflammation inwards. Each division of the ear transmits its disease to a certain region of the brain, the meatus auditorius externus to the lateral sinus and the cerebellum, the tympanum to the cerebrum, and the labyrinth to the medulla oblongata.

Gull* considers that these rules of Toynbee's require the confirmation of further observations, and narrates one of his own cases, where the inflammation extended from the tympanum, but death resulted from an abscess of the cerebellum, which latter depended on a phlebitis of the vena aqueductus vestibuli. Another exception to Toynbee's law is furnished by another case of Gull's, in which an acute inflammation of the lateral sinus resulted from caries of the roof of the tympanum and mastoid. Gull therefore modifies Toynbee's law, and considers that the cerebellum and lateral sinus may suffer from disease of the mastoid, while the cerebrum is endangered by caries of the tympanic roof. In the beginning of a case it would naturally be impossible to prognosticate the course of the disease.

That Gull's modification of Toynbee's law is a just one is shown by other cases. In one case of otorrhœa which fol-

* Guy's Hospital Reports, 3d Series, Vol. III. Meyer, l. c.

lowed measles in childhood, and had existed for forty-three years, a purulent inflammation of the tympanum, with a polypoid growth, caused a thickening of the osseous roof of the tympanum, and not a disease of the cerebrum, as Toynbee would have expected; but the pus was forced through the fenestra ovalis into the labyrinth, and from here followed the auditory nerve through the meatus internus to the cerebellum; the auditory and facial nerves were disorganized by purulent inflammation, and in the left lobe of the cerebellum was an abscess of the size of a walnut, separated from the meninges by a thin layer of brain-substance. The abscess of the cerebellum can result from an otorrhœa without a perforating caries of the bone, both by extension from the labyrinth along the vena aqueductus vestibuli, the auditory or the facial nerve, and also by extension from the mastoid cells. A third possible origin is the posterior wall of the external meatus, as occurred in a case of Hughlings Jackson and Hutchinson*: here a fistula was found between a carious petrous bone and a large abscess in the anterior part of the right cerebellum, the anterior wall of the abscess being formed by the perforated dura mater and the posterior wall by a firm membrane. In this case, an interesting fact was the existence of a smaller encysted abscess directly behind the first, and a larger, not encysted, purulent collection on the inner side and parallel with the other two.

The abscess of the cerebrum in otorrhœa always is caused by a caries of the tympanic roof; often the lamella of bone has been destroyed by suppuration. In one case, the abscess was between the tentorium and the right posterior lobe; but generally it lies deeper in the cerebrum, and is separated from the carious bone by a layer of brain-substance.

Meyer's tables, giving the age at which abscess of the brain was found, and especially showing the influence of otorrhœa, are of special interest:—

* Cases of abscess in the Brain. Medical Times and Gazette, Feb. 23, 1861.

3 between 1-10 years of age. (1 traumatic; 1 otorrhoeal.)					
12	"	10-20	4	"	2
18	"	20-30	2	"	6
11	"	30-49	8	"	3
15	"	40-50	2	"	1
7	"	50-60			1
5	"	60-70			"
<hr/>			17	"	14
71					"

Another of his tables showing the causes of the abscesses is as follows:—

Typhus	1
Intracranial tumor	2
Acute pyæmia	2
Disease of nasal mucous membrane	3
Disease of the blood-vessels	5
Inflammation of neighboring parts of the brain	5
From unknown causes	11
Suppuration of distant organs, especially the lungs	19
Caries of the petrous bone	20
Injuries	21
<hr/>	
Total	89

Hemorrhage within the cranium, due to inflammation of the ear, has never been found except from the lateral sinus, caused by caries of the mastoid and a perforation of the sinus by ulceration. Whether it ever could occur from either of the petrosal sinuses is doubtful, as from their smaller size they would be liable to become closed by the formation of a thrombus before their walls had been perforated. Hemorrhage from the lateral sinus may be either external into the tympanum and meatus, or internal into the cranial cavity. Wreden* has described a case, fatal from two perforations of this sinus, through one of which the hemorrhage was external and through the other internal: he has also given a synopsis of eighteen such perforations of the sinus from various authors.

* Monatschrift für Ohrenheilkunde. No. 10. 1869.

One class of cases, just the reverse of those which we have been considering, remains to be mentioned, namely, that in which the inflammation of the brain is the primary disease, and the inflammation of the ear is secondary. Observations are wanting as yet to show that this ever occurs, except in cerebro-spinal meningitis. In this disease, however, it has been demonstrated, post-mortem, by Moos,* that the purulent inflammation of the meninges may extend along the auditory nerve in the meatus internus, and set up an inflammation of the labyrinth of the ear, or may even extend so far outwards as to set up a purulent inflammation of the tympanum also.

The differential diagnosis between the various forms of brain-disease caused by inflammation of the ear is always difficult, and often impossible, from the fact that meningitis, phlebitis and encephalitis frequently exist together.

Meningitis, in its pure form of inflammation of the pia mater, is never found as the result of ear-disease, but is always associated with, and the result of, an earlier pachymeningitis or inflammation of the dura mater.

Phlebitis of the sinuses is also usually associated with pachymeningitis from the inflammation of the walls of the sinus extending into the dura mater. This, however, is not necessarily the case, for the dura mater may escape inflammation, and the thrombus within the sinus, by breaking up or suppuration, lead to emboli in distant parts, or to purulent infection.

I cannot better show the important part which inflammation of the ear plays in the production of these diseases, and the complicated character of their symptoms, than by the following quotations from Niemeyer †:—

"It is very doubtful whether pachymeningitis externa ever appears as a primary and independent disease, the result of cold or other injury. Certainly, as a rule, it is a secondary affection, associated as such with fissures and fractures, and particularly with caries of the cranial bones, especially of

* Pathological Anatomy of the Ear. Archives of Ophthalmology and Otology. Vol. III., No. 2, page 177.

† Pathologie u. Therapie. Vol. II., page 225.

the petrous and ethmoid bones, or with caries of the upper cervical vertebrae. A pachymeningitis is also sometimes developed in the course of a periostitis of the external cranial surface without one being able to recognize the continuity of the two processes by changes in the cranial bones. An inflammation of a sinus of the brain with resulting thrombus, or a thrombus of the sinus causing eventually an inflammation of its walls, is comparatively common; it occurs most frequently in those sinuses lying in apposition to the petrous bone, the sinus lateralis and the sinus petrosus superior and inferior. This is easily explained, from the fact that an inflammation and thrombus of a brain sinus is caused in the great majority of cases by caries of the petrous bone, which has extended to the base of the skull. The danger of an inflammation and thrombus of these sinuses hangs, like the sword of Damocles, over the numerous patients who suffer from chronic otorrhœa and caries of the petrous bone, the result of an otitis interna. Not infrequently the thrombus passes into purulent degeneration, and, by its particles passing into the evacuant veins, leads to emboli and metastatic inflammations.

"The anatomical changes in light or chronic cases of pachymeningitis externa are confined to a gradual thickening of the dura mater, the result of a growth of connective tissue on its external surface; by this growth the dura mater is firmly attached to the skull; and, later, the layer of newly-formed connective tissue becomes partially ossified. In acute and severe cases the dura mater becomes reddened, thickened, and œdematosus from an injection of the vessels and small ecchymoses, usually on a circumscribed spot corresponding to the seat of injury, or the caries of the bone; later, it is discolored, its tissue becomes loosened and softened, and finally passes into suppuration, which, when the pus collects between the dura mater and the bone, leads to a separation of the inflamed spot from the bone lying beneath. In this latter case the pia mater is almost always likewise inflamed over a large portion of its surface. At an autopsy it is often difficult to distinguish whether the inflammation of the wall of the sinus preceded the thrombus, or

the thrombus the inflammation of the sinus-wall. If the thrombi are not degenerated and broken up, they adhere firmly to the loosened, raw inner surface of the thickened wall of the sinus, and from here extend sometimes backwards to the torcular Horophili, and in some cases downwards to the internal jugular, as Lebert, who has chiefly contributed to the better recognition of this disease, has shown. More frequently, at an autopsy, the thrombus is found already broken up, and the sinus filled with a purulent, sometimes grayish-green, and offensive fluid, mixed with flocculi. In addition to these changes those of an otitis interna and extensive caries of the petrous bone are also found; viz., destruction of the membrana tympani, loss of the ossicula, polypoid growth of the mucous membrane, the tympanum and mastoid cells filled with pus and the labyrinth and cochlea infiltrated.

The chronic pachymeningitis which leads to thickening of the dura mater, its firm adhesion to the skull and ossification of its thickened layers, may be accompanied by headache and other symptoms; but these symptoms are not characteristic and distinctive of the disease. The symptoms and course of an acute pachymeningitis are also so often modified by the symptoms of the primary disease, by the complication of an extended inflammation of the pia mater, and by an extension of the inflammation to the sinuses, that it is impossible to give a clear picture of the disease by itself. If, during an injury to the skull or an otorrhoea dependent on caries of the petrous bone, unusually severe and extensive pain comes on in the neighborhood of the affected bone with febrile disturbance, vomiting, dizziness, subjective noises in the ear, twitchings, delirium, and other symptoms of irritation of the brain, and, later, symptoms of depression and general paralysis, we may assume that the disease of the skull has led to an inflammation of the dura mater, and, later, to diffuse inflammation of the pia mater. The earlier symptoms are often very short and slight, and the patient is found at the first visit, or on his reception at the hospital, in a deep sopor. In such cases, however, the diagnosis can be made with tolerable certainty if an injury to the skull, or,

especially, if a chronic otorrhœa can be discovered, and the other causes of brain disease are wanting."

"The symptoms of inflammation and thrombus of the brain sinuses are always united with the above symptoms of meningitis, as are frequently, also, those of an encephalitis.

"In most cases, only the appearance of chills and of metastatic collections in the lungs allows us to determine with certainty that a caries of the petrous bone has led not alone to a meningitis and encephalitis, but also to the formation of a thrombus in the sinuses."

The symptoms of phlebitis and of thrombosis of the sinuses, which had been insisted upon by Gerhard as pathognomonic of the disease, have been proved by later observations to be of no value. Gerhard laid special weight on the lesser distension of the jugular vein which came from the closed sinus than that of the opposite side; but Schwartz* has described a case in which exactly the opposite condition existed; the jugular on the side in which a large thrombus existed in the longitudinal, lateral and petrosal sinus being more distended than the vein on the opposite side.

Griesinger considered that a painful œdema over the mastoid was diagnostic of the disease, owing to an extension of the thrombus through the vein which leads from the lateral sinus through the foramen mastoideum to the outside of the skull. As will be shown later, the observations of Wreden confirm this as a symptom of phlebitis of the lateral sinus. Schwartz insists, even more strongly than Niemeyer, that the diagnosis of phlebitis and thrombus of the sinuses from disease of the ear can be made with certainty only when pyæmic symptoms are present; *i. e.*, irregularly recurring chills and signs of metastatic deposits in the lungs, spleen, kidneys or joints.

It will be seen from the quotation from Niemeyer that he speaks often of the existence of caries of the bone as the cause of the inflammation of the brain, and would seem to imply that that alone was to be feared. Observations of later years have proved, however, that caries of the bone is by no means necessary for an extension of an inflammation of the ear to the brain; but that numerous channels of com-

* Archiv für Ohrenheilkunde, Vol. VI., page 219.

munication between the ear and brain exist, as shown in the earlier part of this paper, along which a simple inflammation may extend. In three cases of phlebitis and thrombosis of the lateral and petrosal sinuses, due to ear-disease, reported by Schwartze,* one showed no caries of the bone; and in six cases, by Wendt, caries existed in only five.

Wreden† differs from Schwartze and Niemeyer, and claims that the diagnosis of phlebitis and thrombosis of the cerebral sinuses is by no means as difficult and uncertain as has been supposed. He has analyzed one hundred and fifty-one cases by various authors, and points out the necessity of a separation, pathologically, of the different cerebral sinuses, and considers that the little practical use which has been made of our knowledge of inflammation and thrombosis of these sinuses has been due not to the nature of the disease, but to the unscientific use of the cases heretofore reported, in that authors have not properly considered (1) the strict difference between thrombosis and phlebitis; (2) the pathological or, more strictly speaking, the anatomical, differences of the cerebral sinuses.

Thrombosis must be due to mechanical influences which retard the passage of the blood in the sinus, as (1) a diminution of the propulsive force of the heart (marantic form); (2) incomplete emptying of the right heart, in consequence of impeded expansion of the lungs (form due to back pressure); (3) narrowing of the calibre of the sinus in consequence of the pressure of tumors, foreign bodies, etc. (compression thrombosis); (4) coagulations in a number of the afferent or large efferent veins (by extension). It is not accompanied by fever, and produces no pyæmic symptoms.

Phlebitis, however, arises (1) from the propagation of inflammatory processes (*per contiguitatem*) from the vicinity of sinuses to their own walls; (2) from direct traumatic injury of the walls; (3) by transference of the phlebitic process (*per continuatatem*) from single large veins that communicate with it. It is accompanied by violent fever,

* Loc. cit.

† Saint Petersburger Medicinische Zeitschrift, Vol. XVII.; Archives of Ophthalmology and Otology, Vols. IV. and V.

and very often gives rise to pyaemic or even septicaemic symptoms.

The history of the case with the predisposing disease, if any existed, but more particularly the violent fever and the pyaemic symptoms, render the differential diagnosis between the two conditions comparatively easy.

Thrombosis of the cavernous sinus shows itself by a congestion of the vein which empties into it, the ophthalmic, and its branches; these branches are the supra-orbital, muscular and lachrymal, ciliary, anterior and posterior nasal, ethmoid, frontal and infra-orbital. As the result of the congestion of these veins, there is, on the affected side, edematous swelling of the nostril, forehead and eyelid, mechanical hyperaemia of the retina, with diminution of vision, and also swelling of the nasal mucous membrane with bloody discharge. If a phlebitis of the cavernous sinus exists, in addition to the above symptoms of congestion from the thrombus, which would almost necessarily be present, there are also the phenomena of irritation and paralysis of the abducens, the ophthalmic branch of the fifth and the oculo-motorius nerves, due to the swelling of the walls of the sinus and of the neighboring tissues which press upon these nerves. The affection of the abducens which lies close to the outer wall of the sinus shows itself by paresis of the external rectus, causing an internal squint; that of the ophthalmic branch of the trifacial, which lies close to the sinus below and outwards, by headache especially in the forehead and over the eye (supra-orbital nerve), epiphora (lachrymal nerve) and photophobia (reflex irritation or hyperesthesia of the optic nerve); that of the oculo-motorius, which lies over the upper and outer wall of the sinus, by paralysis of the upper eye-lid with inability to open the eye (ptosis).

Thrombosis of the superior longitudinal sinus shows itself by repeated violent hemorrhages from the nose, due to the back pressure in the veins of the nasal cavity which empty into this sinus; and also by epileptiform convulsions, with loss of consciousness, which Wreden refers to capillary hemorrhages in the cortical substance of the convexity of both

posterior cerebral lobes, brought on by the interference with the blood-current from the surface of the brain. The hemorrhage from the nose, Wreden does not consider important in itself, but in connection with the epileptiform convulsions it is very significant. From an analysis of the recorded cases of thrombosis of the superior longitudinal sinus, he finds that all which presented after death hemorrhages in the cortical substance, as described above, were subject, during life, to epileptiform attacks.

Thrombosis and phlebitis of the transverse sinus show themselves by enormous oedematous swelling of the soft parts in and about the external ear, which has exactly the character of phlegmasia alba dolens: there is also apt to be constant dizziness, even in the horizontal position, and staggering. As the phlebitis extends downwards to the internal jugular vein, the external phlegmonous inflammation spreads from the neighborhood of the mastoid downwards to the clavicle: the point of greatest swelling is along the course of the vein, and there is great tenderness on pressure. As the circulation in the internal jugular vein becomes impeded, symptoms of congestion of the facial vein show themselves by a puffy and swollen condition of the face, which, however, may be only transitory; for the facial vein has so many anastomoses with the branches of the external jugular that a collateral circulation is very soon established. If the phlebitis extends up into the facial vein, its larger branches may become plugged, and then will there be enormous oedema of the face; if the inflammation extends into the finer facial branches, a distinct erysipelatous inflammation, with redness, heat and vesicles, is developed in the skin of the cheek and forehead.

With phlebitis of the internal jugular vein there is a remarkable dilatation of the external jugular, with a distinct undulatory increase and diminution in fulness, accompanying respectively expiration and inspiration. This phenomenon is usually only temporary, and depends on the blocking of the collateral circulation.

Clonic and tonic spasms of the sterno-cleido-mastoid and trapezius muscles may result from an irritation of the spinal

accessory nerve in the jugular foramen when pressed upon by a thrombus of the bulb of the jugular vein.

Thrombosis and phlebitis of the superior petrosal sinus cause great congestion in the labyrinth of the ear, with subjective noises, deafness, and especially a great diminution or total loss of perception of sound through the bones (bone-conduction), due to the fact that the veins of the labyrinth drain, at least partially, into this sinus.

From the direct connection of one sinus with another, it would not be expected that any one set of symptoms would alone be observed.

One important fact in regard to the symptoms of meningitis and phlebitis caused by the ear, although mentioned by Niemeyer in the preceding quotation, deserves to be particularly emphasized,—the earlier and more acute symptoms are often of such short duration, or so slight, that they can escape observation.

Meningitis may, and often does, exist alone, uncomplicated by any other form of brain-disease; phlebitis of the sinuses is almost always accompanied by more or less meningitis, while encephalitis, although it may be complicated by meningitis, or by both meningitis and phlebitis, is frequently found without either of these complications.

The obscurity of the symptoms in abscess of the brain is well known, as is also the marked disproportion between the anatomical changes found after death and the clinical symptoms observed during life. This can be explained, at least in a great measure, by the slow growth of the purulent collection, its softness, which consequently produces slight pressure on the healthy portions of the brain, and the fact that the position of the abscess is usually in the white substance of the hemispheres, which plays a comparatively unimportant part in the physiology of the brain.

In abscess of the brain there is also frequently a marked latency in the disease intervening between the initiatory symptoms and the final, fatal ones; total latency of the disease throughout its whole course is, however, rare, but two cases being reported by Meyer. The primary symptoms seem to mark the early period of congestion and begin-

ning softening, while the final ones are found when the abscess has reached a motor centre, or ruptures, or sets up another disease of the brain, as meningitis or extensive œdema.

The beginning of an encephalitis, always difficult to define, is especially so when caused by disease of the ear, for the pain and other symptoms in the ear, although not necessarily present in a serious ear-disease, are apt to obscure the brain symptoms. The more gradual the extension of the ear-disease to the brain, the slower is the primary congestion of the encephalitis, and the less marked the brain symptoms. Slight headache is often the only brain symptom complained of, but as this is a not infrequent accompaniment of the ear-disease, but little reliance can be placed on it unless it becomes very severe, and long continued, without symptoms of meningitis. In two-thirds of Meyer's cases of abscess of the brain, headache, variable but gradually increasing with the length of the disease, was the first symptom noticed; this was sometimes accompanied with fever, dizziness, and occasionally with vomiting, but seldom with convulsions or paralysis. After headache, fever with chills, thirst and loss of appetite would seem to be the next frequent initiatory symptom. Meyer found it in one-eighth of his cases, while convulsions were the first symptom in one-tenth of his cases. The convulsions were usually general, of an epileptic character, and accompanied by loss of consciousness. Neuralgia, anaesthesia, and disturbances of the intellect are rare as early symptoms.

The less severe the initiatory symptoms of encephalitis, the slower, as a rule, is the development of the abscess. The earlier symptoms often disappear entirely, so that for a time the patient is absolutely free from symptoms of any kind. The course of the disease, after the first symptoms, may be either acute or chronic, depending on whether the abscess becomes encapsulated or not: in the acute form, death results from the increasing suppuration and destruction of the brain-substance; in the chronic form, from rupture of the abscess into the ventricles, or upon the surface of the brain, or else from œdema or anemia.

In the acute abscess there is little, if any, latency in the disease; at the most, the patient is free from symptoms for only a few days, and the disease runs its course and ends fatally in from one to five weeks; occasionally the period is longer. Meyer found the following as the causes of fourteen such acute abscesses: injury, 6; metastasis, 3; emboli, 2; otitis, 2; disease of the nose, 1; showing that no one cause specially predisposes to this form of the disease.

The chronic abscess is characterized by a period of marked latency intervening between the earlier and the later symptoms; this latency is often complete, but is sometimes interrupted by short, remittent attacks of headache. Sometimes the earlier symptoms are wholly overlooked, and the disease runs its course absolutely without noticeable symptoms till the final ones set in. The duration of this latency may vary from a few weeks to several months; and in two cases, narrated by Bruns and Schott, it was thought to have reached twenty years. The period of latency being once disturbed, the final course of the disease is usually rapid. In thirty-four cases given by Meyer, in which the period of latency and that of the final symptoms was well marked, the latter ran their course and ended fatally:—

Within the 1st week	13
" 2d "	7
" 3d "	4
" 4th "	4
" 5th "	4
" 6th "	1
" 9th "	1

Occasionally an abscess without an enveloping capsule assumes this chronic course; but this is rare.

In the chronic abscess, death is usually caused from a rupture into the ventricles, or upon the surface of the brain, from an extensive oedema of the brain-substance, from involvement of the medulla oblongata in the disease, or occasionally from anaemia of the brain. Of these different causes, the rupture into the ventricles is the most frequent,

and is followed by symptoms of irritation in the motor centres, febrile disturbance, delirium, limited or general paralysis of motion and sensibility, coma and death. Rupture on to the base of the brain is generally followed so rapidly by profound coma that other symptoms are masked ; but sometimes headache, dizziness and vomiting are noticed. Hemiplegia and continuous coma have been noticed from extensive œdema ; but the symptoms of all of the above enumerated causes of death are so little distinctive that a differential diagnosis is usually impossible. They all lead to a fatal termination in from a few hours to a few days.

Although the complicated character of the various forms of brain-disease should be borne in mind, still we have many symptoms which enable us to distinguish one disease from the other. In meningitis the following symptoms are usually observed : violent fever, with a very high pulse, especially in the beginning of the attack ; severe headache, general and never distinctly circumscribed ; violent delirium ; contractions of the pupils, and vomiting ; convulsions and sopor.

In encephalitis the diagnosis must be very uncertain, as there may be absolutely no symptoms, or—only in the beginning of the disease—symptoms which resemble, and often are due to, meningitis. When the abscess is in certain positions, there may be affections of the sensible or motor nerves, showing themselves as anaesthesiae or paralyses. The early history will often help the diagnosis, if it shows the existence of a predisposing cause to encephalitis, such as injury or disease of the ear, and especially if there has been an acute illness with meningitic symptoms. The history of an acute febrile attack, resembling meningitis, with a high pulse and severe general headache, followed by a marked falling of the pulse below the normal, and occasional headache, distinctly circumscribed and in one spot, is often characteristic of encephalitis.

Simple thrombosis of the brain sinuses, from ear-disease, is very rare, as all the conditions predispose to a phlebitis, and not to any mechanical obstruction to the circulation ; it can be distinguished from phlebitis by presenting the symptoms of obstruction to the circulation, as described above,

unattended by high pulse and increased temperature; while a phlebitis of the same sinuses would show, in addition to the same obstruction to the circulation, marked febrile disturbance. The occurrence of pyæmic chills and metastatic deposits in the lungs, spleen or liver, render the diagnosis of phlebitis certain.

